patients would need to be treated to prevent one episode of PE or DVT. Serious adverse events were 0.7% with fondaparinux, and 1.1% with placebo, with major bleeding occurring in one patient in each group.

Comment: The number needed to treat, based on relative risk reduction, demonstrated in the trial to prevent one episode of PE with fondaparinux in patients with SVT is 300. This is similar to the number needed to treat with low-molecular-weight heparin compared with placebo or no treatment in trials of VTE prophylaxis in acutely ill medical patients (345, Dentali F, Ann Intern Med 2007;146:278-88). Treatment with fondaparinux also reduced the risk of symptomatic recurrence of SVT and extension to the saphenofemoral junction. Because both of these events may result in escalation of therapy, these end points are clinically relevant. Overall, the data indicate that with pharmacologic therapy it is possible to improve the natural history of SVT. Cost effectiveness and effects on quality of life of the 45-day treatment regimen of fondaparinux for SVT will need to be evaluated both with respect to the cost of fondaparinux and the cost of diagnostic testing to determine eligibility for treatment.

Long-Term Outcome of Symptomatic Severe Ostial Vertebral Artery Stenosis (OVAS)

Karameshev A, Schroth G, Mordasini P, et al. Neuroradiology 2010;52: 371-9.

Conclusion: Vertebral artery stenting can be performed with minimal periprocedural complications. Potential benefits are most likely in those with bilateral vertebral artery stenosis.

Summary: About 25% of all strokes are posterior circulation strokes (Bogousslavsky J, Stroke 1988;19:1083-92). Of posterior circulation strokes, 20% to 25% are associated with a severe ostial stenosis or occlusion of the vertebral artery (Wityk RJ et al, Arch Neurol 1998;55:470-478). Endovascular management of vertebra artery stenosis is infrequently reported. The only randomized trial, The Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS, Coward LJ, Stroke 2007;38: 1526-1530), enrolled only 16 patients with vertebral artery stenosis. An additional trial, the Vertebral Artery Stenting (VAS) trial, may be underway (Trials 2008;9:65). Results are unlikely to be available for many years.

In this study by Karameshev et al, 39 consecutive patients with a recently symptomatic (≤48 hours) severe (>70%) ostial vertebral artery stenosis were evaluated in a single-institution study. Treatment of patients was medical (n = 29) or by vertebral artery stenting (n = 10) and was left to the discretion of the treating physician. Mean follow-up was 2.8 years. Data were analyzed using the Kaplan-Meier method for risk of recurrent stroke, transient ischemic attack (TIA), or death. Stented patients were younger and more likely to have bilateral vertebral artery disease ($P = .04$ and $P = .02$). Vertebral artery stenting was successful in all 10 patients. One patient had a periprocedural transient ischemic attack. There was restenosis of one vertebral artery stent at 9 months. At 4 years of follow-up there was no statistical difference in the risk of a combined end point of TIA and stroke in the posterior circulation in stented (10%) compared with medically treated patients (45%; relative risk, 0.24; 95% confidence interval, 0.031-1.85; $P = .095$). Patients with bilateral vertebral disease had 0% recurrence of symptoms after stenting at 4 years compared with 91% for medical treatment (relative risk, 0.10; 95% confidence interval, 0.022-0.49; $P = .004$).

Comment: The study was not randomized and the patients were highly selected for treatment. Clearly, the study does not provide sufficient data to justify a widespread approach to endovascular management of vertebral artery stenosis. Medical management remains the mainstay for patients with symptomatic vertebral artery stenosis with intervention by open or endovascular techniques reserved for highly selected patients using what amounts to really no more than "best guess" criteria.